Crysler, Ruby

From:	Kidwell, JessicaL
Sent:	Friday, November 18, 2016 2:43 PM
To:	Crysler, Ruby
Subject:	RE: McConnell AFB PBR: RTC: SS544 (SWMU 207) Draft RFI Report
Attachments:	EPAR7 2016 Less-Than-Lifetime TCE Risk Characterization and Action Levels_November 2016-Final.pdf
Categories:	Record Saved - Shared

Hi Ruby:

Thanks for your emails. The following responses are provided for your consideration (see column added at right). If you haven't already, please also consider sharing our November 2, 2016, memorandum: "EPA Region 7 Action Levels for Trichloroethylene in Air" (attached).

As an insufficient heads up … I'm here until 3:15 today if you have questions or concerns. However, I'm out all next week, returning 11/28/16.

Enjoy your holiday, Jesse

Section 6.1, page 6-1 (Receptors and Exposure Pathways): The section states, "Based on current groundwater concentrations, surface conditions (predominantly paved and open-air), and the general absence of any structures (with the exception of the Control Tower), exposure via vapor intrusion or inhalation of CVOCs from groundwater at SWMU No. 207 is incomplete." The following issues are noted:			
a. Although the Control Tower is not a residential building, the EPA has broad authority and distinct responsibilities to assess and, if warranted, mitigate vapor intrusion in non-residential settings arising from a chemical release that causes subsurface contamination by hazardous, vapor-forming chemicals (EPA, 2015a). The Control Tower is an occupied building and should not be excluded from vapor intrusion assessment.	D	The current Control Tower is in the process of being decommissioned. A new Tower, currently in the design phase, is being built which will incorporate an engineered vapor barrier in the foundation. Estimated completion date for the new tower is March 2019. For the current configuration of the control tower, all workers are located on the second floor of the building, limiting their exposure to the vapor intrusion pathway.	Cu de ex he As to de ur ca m

b. Although no shallow groundwater or soil gas sample has been collected near the Control Tower and no building configuration is available, current TCE concentrations in groundwater may pose a vapor intrusion concern to the Control Tower. Using the Vapor Intrusion Screening Level calculator (EPA, 2015b), an indoor air concentration of 7.43 micrograms per cubic meter is estimated based on a groundwater concentration of 26 µg/L (MW-179), a groundwater temperature of 18°C, and a commercial exposure scenario. The EPA Region 7 worker action level, based on potential fetal cardiac defects, is 6 µg/m^3 for an acute exposure of 8 hours.	D	If the "Exposure Scenario" in the VISL Calculator is changed to commercial and the generic attenuation factor for source medium of vapors for groundwater is changed to 0.0005 (from EPA's Vapor Intrusion Guidance, June 2015, for soils where groundwater is below fine-grained vadose zone soils, when laterally extensive layers are present) under Commercial in number 2 of the Notes, the 26 ug/L value has a carcinogenic risk of 1.2x10-6 and a hazard index of 0.42, both of which are within acceptable ranges.	M gr ex te (E Hc in ta lif pr in ai fo be Ac sit ge 10 gr bu pa m Fc 50 cc cc th to
c. Although the Control Tower appears to be the only occupied building within the SWMU 207 boundary, occupied buildings are present downgradient of SWMU 207 and are underlain by chlorinated volatile organic compound plumes of sufficient concentration to pose vapor intrusion concern. Therefore, additional assessment of the vapor intrusion pathway, using multiple lines of evidence, is warranted at this site.	D	Response to comments #25 and #26 address the current Control Tower. The authors would agree that the contaminant plume associated with the former Boeing North Hangar warrant an assessment of the vapor intrusion pathway. However, based on the analysis described in **Section 5.3 and 5.4** of the RFI, this plume is related to former activities on that property and do not initiate from SWMU 207. Therefore the North Hangar will not be included in the SWMU 207 Baseline Risk Assessment.	Tc cc bu w. su fu dc er pl in

Appendix D (Slug Test Analysis): The plots of normalized head data versus time for wells MW-49D, MW-50D, MW-178 and MW-180 are concave upward, a curvature that can make analysis by straight-line methods such as Bouwer and Rice (1976) ambiguous. Butler (1998) recommends matching Bouwer and Rice (1976) solutions to data within a normalized head range of 0.20 to 0.30 to minimize ambiguity associated with data curvature, and improve reliability of the data analysis. The employed slug test analysis software, AQTESOLV, is capable of superimposing recommended normalized head ranges on data plots to enhance visual curve matching. It is recommended that normalized head range be used or GSI should select an alternative analytical model appropriate for the formation and well installation.	D	The slug test analysis was performed using the Butler (1998) consideration of normalized head as described by the reviewer. The straight-line visual matching was performed over the normalized head range of 0.2 to 0.3, as is shown on the graphs included in **Appendix D**. However, as suggested by the reviewer, the normalized head ranges used for curve matching will be superimposed on the graphs in order to aid in review of the analysis.	Th gr

From: Crysler, Ruby
Sent: Friday, November 18, 2016 8:57 AM
To: Kidwell, JessicaL <Kidwell.JessicaL@epa.gov>
Subject: RE: McConnell AFB PBR: RTC: SS544 (SWMU 207) Draft RFI Report

Jesse,

Thanks for looking at the responses. Are you ok with their discussion about VI assessment at the air control tower?

Section 6.1, page 6-1 (Receptors and Exposure Pathways): The section states, "Based on current groundwater concentrations, surface conditions (predominantly paved and open-air), and the general absence of any structures (with the exception of the Control Tower), exposure via vapor intrusion or inhalation of CVOCs from groundwater at SWMU No. 207 is incomplete." The following issues are noted:		
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Phone: 913-551-7409

From: Kidwell, JessicaL
Sent: Tuesday, October 11, 2016 12:00 PM
To: Crysler, Ruby <Crysler.Ruby@epa.gov>
Subject: RE: McConnell AFB PBR: RTC: SS544 (SWMU 207) Draft RFI Report

Hi Ruby:

Thanks for sharing these with me. In general, the responses are acceptable; however, the following items may warrant a little clarification.

- Items 5 and 10 - E. Section 2.3 will be revised to note the change in monitoring wells sampled. Less clear is whether Section 2.3 will be revised to discuss the basis for the replacement well locations or the historical groundwater analyses for hexavalent chromium at SWMU 207. These aspects of the response should be included in the report.

- Item 26 – D.
    - Modification of the generic attenuation factor is appropriate, so long as the justification points to specific evidence of laterally-extensive, fine-grained soils beneath the building basement or foundation.
    - Using the modified groundwater-to-indoor air attenuation factor (0.005) and a site-specific groundwater temperature (18°C), the calculated indoor air TCE concentration is 3.71 $\mu g/m^3$. This calculated indoor air TCE concentration is below the EPA Region 7 worker action level of 6 $\mu g/m^3$ based on an 8-hour exposure period. (Note that the facility continues to evaluate TCE vapor intrusion based on the target cancer risk and hazard quotient. EDAB hopes to share an R7 technical memorandum on the acute risks of TCE in air this week; action levels are specified within.)

- Items 14 and 27 – D. Please note these responses, which attribute responsibility to Boeing.

Let me know if you have questions.

Thanks again, Jess

From: Crysler, Ruby
Sent: Wednesday, October 05, 2016 2:05 PM
To: Kidwell, JessicaL <Kidwell.JessicaL@epa.gov>
Subject: FW: McConnell AFB PBR: RTC: SS544 (SWMU 207) Draft RFI Report

Jesse,

McConnell response to EPA comments on the Draft SWMU 207 RFI report are attached. Please review them when you have time and let me know if their responses are satisfactory.

Thank you.

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From: Wight, Brian [mailto:brian.wight@aecom.com]
Sent: Wednesday, October 05, 2016 1:46 PM
To: Crysler, Ruby <Crysler.Ruby@epa.gov>
Cc: Jacqueline Grunau (jgrunau@kdheks.gov) <jgrunau@kdheks.gov>; Mark D. Wichman (mark.d.wichman@usace.army.mil) <mark.d.wichman@usace.army.mil>; Sansom, Andrea NWO <Andrea.Sansom@usace.army.mil>; KNIGHT, COLE D GS-11 USAF AMC 22 CES/CEAN (cole.knight@us.af.mil) <cole.knight@us.af.mil>; BLAIR, SHELDON M CTR USAF AMC 22 CES/CEIE <sheldon.blair.ctr@us.af.mil>; Krause, Michael <michael.krause@aecom.com>; Mike L. Schofield (mlschofield@gsi-net.com) <mlschofield@gsi-net.com>; Bergantzel, Vanessa <Vanessa.Bergantzel@aecom.com>; Julie Spencer <jaspencer@gsi-net.com>
Subject: McConnell AFB PBR: RTC: SS544 (SWMU 207) Draft RFI Report

Ruby,

URS/GSI responses to EPA's comments on the SS544 (SWMU 207) Draft RFI report are attached for your review and approval. If possible, please provide your approval on or before 14 October 2016. If this is not possible, please let us know when your approval may be received.

Thanks

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NOV 0 2 2016

MEMORANDUM

SUBJECT: EPA Region 7 Action Levels for Trichloroethylene in Air

FROM: Mike Beringer, Chief MB
Environmental Data & Assessment Branch
Environmental Sciences & Technology Division

TO: Branch Chiefs
Waste Enforcement and Materials Management Branch
and Waste Remediation & Permitting Branch
Air and Waste Management Division

Branch Chiefs
Superfund Division

The purpose of this memorandum is to update the U.S. Environmental Protection Agency Region 7 RCRA and Superfund programs on the recommended action levels for trichloroethylene (TCE) in air, and provide information on characterizing and addressing human health risks from less-than-lifetime exposures. The action level for a residential scenario is 2 $\mu g/m^3$, and the action level for an industrial/commercial scenario with an 8-hr workday is 6 $\mu g/m^3$. Equations to allow derivation of action levels for alternative scenarios, such as a 10-hr workday, are presented. As described in this attachment, it is assumed that an exposure to TCE at any time during an approximate three-week period in early pregnancy could result in one or more types of cardiac malformations. Thus, the critical exposure period of concern used to evaluate the potential for heart defects and derive action levels for TCE is one day. An exceedance of the TCE action level indicates a potential imminent threat to human health. Region 7 should expedite early or interim action(s) to eliminate, reduce, and/or control the hazards posed by the site as quickly as possible. If you or your staff have any questions or need further assistance, please contact Kelly Schumacher (x7963).

EPA Region 7 Action Levels for Trichloroethylene in Air.	
Exposure Scenario	*Action Level*
Residential (24 hours/day)	**2 $\mu g/m^3$**
Industrial/Commercial (8 hours/day)[1]	**6 $\mu g/m^3$**

[1] Site-specific action levels should be derived when the workday differs from 8 hours/day.

Attachment

EPA Region 7 Action Levels for Trichloroethylene in Air

Introduction

In 2011, the latest human health toxicity values for trichloroethylene were published by the United States Environmental Protection Agency's Integrated Risk Information System program (EPA, 2011a). As discussed in this document, these new values are partly based on developmental health effects that result from less-than-lifetime exposures. In contrast, the toxicity values typically used to evaluate potential health risks and derive action levels at Superfund and RCRA sites are based on health effects associated with long-term, or chronic exposures. Further, the equations and exposure parameters used typically reflect all or a significant portion of a person's lifetime. Once the current TCE values were released, the protectiveness of using traditional approaches to assess and address TCE exposures was questioned. The purpose of this memorandum is to update the EPA Region 7 RCRA and Superfund programs on the recommended action levels for TCE in air and provide information on characterizing and addressing human health risks from less-than-lifetime exposures. To support these objectives, the window of susceptibility for the developmental toxicity associated with TCE is examined, the critical exposure period of concern is identified, and the appropriate exposure parameters and equations are elucidated.

Toxicity Assessment

The EPA's final toxicological review by the IRIS program incorporates comments by the U.S. National Academy of Sciences (National Research Council, 2006), two U.S. EPA Science Advisory Boards (EPA, 2002 and 2011b), the Executive Office of the President (Office of Management and Budget, 2009 and 2011), the U.S. Department of Defense (DOD, 2009a, 2009b and 2011), the National Aeronautics and Space Administration (NASA, 2009 and 2011), internal Agency reviewers, and the public, among others. The Halogenated Solvents Industry Alliance, Inc., which represents the interests of TCE manufacturers and producers, submitted a Request for Correction of the TCE IRIS assessment (HSIA, 2013), which was denied by the EPA's Acting Assistant Administrator (EPA, 2015). The HSIA then submitted a Request for Reconsideration (HSIA, 2015), which was also denied by the EPA (EPA, 2016a). The EPA found the Requests "directly contrary to the SAB's conclusions and recommendations, such that to accept HSIA's RFC/RFR would require EPA to reject SAB's advice" (EPA, 2016a).

The EPA's Office of Land and Emergency Management recognizes an IRIS assessment as the official Agency scientific position regarding the toxicity of a chemical based on the data available at the time of the review (EPA, 2003). As such, IRIS is generally the preferred source of human health toxicity values used to evaluate risks at Superfund and RCRA hazardous waste sites. In accordance with Directive 9285.7-53 (EPA, 2003), the 2011 IRIS TCE toxicity values will be used to evaluate risks and derive action levels by the Region 7 RCRA and Superfund programs until the 2011 values are either revised or rescinded.

Non-Carcinogenic Health Effects

In general, the EPA assumes that a dose or exposure level exists below which adverse non-carcinogenic health effects will not occur (EPA, 1989). Below this threshold, it is believed that exposure to a chemical is tolerated without adverse effects. Adverse health effects occur only when physiologic protective mechanisms are overcome by exposure to doses or concentrations above the threshold. For chronic toxicity values, the first adverse effect (or its known precursor) that occurs to the most sensitive species as the dose rate of an agent increases, regardless of the exposure duration, is designated the critical endpoint. The dose or exposure at which the critical endpoint is observed is the point of departure. Uncertainty factors, ranging from 1 to 3,000, reflecting limitations of the data used are applied to the point of departure to derive the inhalation reference concentration. The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 1989).

The 2011 Scientific Advisory Board panel recommended that, "The two endpoints for immune effects from Keil *et al.* (2009) and the cardiac malformations from Johnson *et al.* (2003) should be considered the principal studies supporting the RfC" (EPA, 2011b). The panel considered the immune effects and cardiac malformations co-critical endpoints (EPA, 2011b). In accordance with the SAB panel recommendations, the IRIS program based the TCE chronic reference concentration of 2 $\mu g/m^3$ on these two co-critical endpoints, each of which can support the RfC independently: autoimmune disease following chronic exposure in adults (0.00033 ppm, or 1.8 $\mu g/m^3$) and heart defects following exposure during early pregnancy (0.00037 ppm, or 2.0 $\mu g/m^3$). The RfC is also supported by nephrotoxicity (kidney effects) following chronic exposure in adults (0.00056 ppm, or 3.0 $\mu g/m^3$). Following publication of these values, the developmental cardiac effects were further addressed by the IRIS program in "TCE Developmental Cardiac Toxicity Assessment Update" (EPA, 2014a) and by scientists in the EPA's Office of Research and Development in the peer-reviewed literature (Makris *et al.*, 2016).

Chronic exposure to TCE poses a potential human health hazard to the central nervous system, kidneys, liver, immune system, and male reproductive system. As mentioned above, immunotoxicity in adults is considered a co-critical endpoint, at a slightly lower concentration than that associated with cardiac defects. Overall, the IRIS program concluded that "the human and animal studies of TCE and immune-related effects provide strong evidence for a role of TCE in autoimmune disease and in a specific type of generalized hypersensitivity syndrome" (EPA, 2011a). Kidney toxicity was considered a supporting endpoint, with high confidence found in multiple lines of evidence in both human and animal studies.

Short-term exposures to TCE during pregnancy are associated with many forms of developmental toxicity, including spontaneous abortions, decreased growth, developmental neurotoxicity, developmental immunotoxicity, and birth defects. However, the critical developmental endpoint is cardiac malformations. The primary types of heart defects observed with TCE exposures include atrial and ventricular septal defects, which are holes in the wall (septa) between the top two chambers (atria) or bottom two chambers (ventricles) of the heart, and pulmonary and aortic valve stenoses, which are thickened or fused heart valves that do not properly open and/or close and may leak blood. The critical window of susceptibility for these types of defects is an approximate three week period (i.e., valvuloseptal morphogenesis, or the period in which major cardiac morphogenic events such as heart valve formation occur) approximately four to seven weeks after conception, early in the first trimester of human pregnancy (Dhanantwari *et al.*, 2009). The type and severity of the resulting cardiac malformation or malformations depends on the timing and level of exposure to TCE within this approximate three week period. Exposures that clear the body before this period do not impact the heart valves and septa, because they have not yet begun to form. In humans, TCE and most of its metabolites are eliminated within a week of exposure (EPA, 2011a).

Carcinogenic Effects

The EPA evaluates carcinogenicity in two parts (EPA, 2005a). First, the Agency evaluates all available scientific information and assigns a weight-of-evidence classification based on a compound's potential to cause cancer in humans. In the absence of sufficient data regarding the mode of action or if the weight-of-evidence supports a mutagenic mode of action, the EPA generally assumes that any exposure to a chemical will increase an individual's risk of developing cancer. Under this default approach, there is no threshold below which the probability of developing cancer is zero. Second, a toxicity value is derived to define the quantitative relationship between dose or concentration and carcinogenic response. For inhalation exposures using the default approach, this value is known as the inhalation unit risk. The IUR is a generally plausible upper-bound estimate of the increased probability of developing cancer following a lifetime of exposure. This value is used to estimate the increased risk of developing cancer from inhalation of potentially carcinogenic chemicals.

Following the EPA's Guidelines for Carcinogen Risk Assessment (EPA, 2005a), the IRIS program has evaluated the carcinogenic potential of TCE and has classified it as "carcinogenic to humans" by all routes of exposure. This conclusion is based on convincing evidence of a causal association between TCE exposure in humans and kidney cancer, strong evidence of non-Hodgkin's lymphoma, and more limited evidence of liver and biliary tract cancer. The inhalation unit risk for TCE, based on these combined cancer types, is 4.1E-06 $(\mu g/m^3)^{-1}$. Sufficient evidence supports a mutagenic mode of action for TCE-induced kidney tumors in humans, but modes of actions have not been established for the other TCE-induced cancer types. The portion of the TCE IUR specific for kidney tumors is 1.0E-06 $(\mu g/m^3)^{-1}$, while the IUR for non-Hodgkin's lymphoma plus liver and biliary tract cancers is 3.1E-06 $(\mu g/m^3)^{-1}$.

Risk Characterization

The EPA's RCRA and Superfund programs characterize potential human health risks using standardized equations that combine toxicity values with exposure parameters because risk is a function of both hazard and exposure. Typically, the EPA's standard default exposure parameters for chronic scenarios, published in OSWER Directive 9200.1-120 (EPA, 2014b), are used. However, exposure assessments must take into account the time scale related to the specific biological response (NRC, 1991). This means that exposure parameters selected to evaluate risks and/or develop levels of concern for a given chemical and scenario should correspond as closely as possible with the exposure period used to develop the toxicity value. For example, time-weighted average exposures over a lifetime have little relevance for a developmental toxin if the adverse effects could only occur following exposure during a particular stage of development (EPA, 1992).

Non-Cancer Hazard Quotients for Cardiac Defects

The toxicity values considered protective for a lifetime of exposure to TCE are partly based on non-cancer health effects resulting from less-than-lifetime exposures. As previously stated, one of the two co-critical endpoints that serves as the basis for the TCE RfC is cardiac defects. This effect can only occur when the fetus is exposed during the period of heart development. Therefore, the EPA's standard default exposure parameters for chronic exposures are invalid for estimating hazard quotients representing the potential for cardiac defects associated with TCE exposures and for deriving TCE levels of concern that are protective of developmental endpoints. To select appropriate less-than-lifetime exposure parameters that may be used to characterize these hazards and derive levels of concern, the critical exposure period of concern for TCE-related heart malformations must first be identified.

"[F]or developmental toxic effects, a primary assumption is that a single exposure at a critical time in development may produce an adverse developmental effect, i.e., repeated exposure is not a necessary prerequisite for developmental toxicity to be manifested" (EPA, 1991). The EPA's Risk Assessment Guidance for Superfund Part A (EPA, 1989) directs the use of a day or a single exposure incident to assess the potential risks of adverse developmental effects. Following this guidance, it is assumed that a single exposure to TCE at any time during the approximate three week period of valvuloseptal morphogenesis could result in one or more of the types of heart malformations described previously. Thus, the critical exposure period of concern used to evaluate the potential for cardiac defects is one day. A 24-hour exposure period has been used by the EPA to evaluate acute hazards associated with TCE in the final, peer-reviewed TSCA Work Plan Chemical Risk Assessment (EPA, 2014c).

The EPA's Risk Assessment Guidance for Superfund Part F, Supplemental Guidance for Inhalation Risk Assessment (EPA, 2009) specifies that the exposure concentration (EC) that should be used to evaluate risks and derive levels of concern for acute endpoints is equivalent to the concentration detected in air (CA), as shown in Equation 1.

$$EC\left(\frac{\mu g}{m^3}\right) = CA\left(\frac{\mu g}{m^3}\right) \tag{1}$$

For a residential scenario, in which exposure to TCE inside a home is assumed to occur throughout the entire exposure period of concern, Equation 1 is appropriate. However, for other types of scenarios (e.g., industrial, commercial, recreational), exposures to TCE only occur for a portion of any given 24-hour period. Moreover, exposures to different concentrations of TCE may occur within a single day at some sites. To account for these multiple exposures, Equation 1 can be modified, resulting in a time-weighted average exposure concentration. The 24-hour TWA exposure concentration can be calculated using Equation 2.

$$EC_{24} = \sum_{i=1}^{n}(CA_i \cdot ET_i)/\,AT_{24} \tag{2}$$

where: EC_{24} ($\mu g/m^3$) = time-weighted average exposure concentration over 24 hours;
CA_i ($\mu g/m^3$) = TCE concentration in air in microenvironment (ME) i;
ET_i (hours) = exposure time spent in ME i;
AT_{24} (hours) = averaging time for the exposure period of concern (24 hours)

In a residential scenario, there is a single microenvironment, the residence, with an exposure time of 24 hours. Thus, the Residential EC_{24} will equal CA_{res}, as shown in Equation 3. To reduce uncertainty in residential scenarios, CA_{res} should be based on air samples collected for an entire 24-hour exposure period. Generally, stationary 24-hour indoor air sample results are used.

$$Residential\ EC_{24} = \frac{(CA_{res} \cdot 24\ hrs)}{24\ hrs} = CA_{res} \tag{3}$$

In a typical industrial or commercial scenario, there are two microenvironments. One is the workplace, and the other is away from the workplace. The Industrial/Commercial EC_{24} can be calculated using Equation 4, below. Although the standard value for ET_{work} is an 8-hour workday, this variable should reflect site-specific conditions. For example, employees at a given site may work longer shifts, such as 10 or 12 hours, and they may or may not take their lunch breaks on site. CA_{work} should be based on air samples collected for the entire exposure time, ET_{work}, during the portion of the day that workers are present. This is to prevent potential underestimates of TCE concentrations if diurnal variations occur at a site, although such variability does not exist at all sites. Generally, stationary 8-hour or 10-hour indoor air samples are appropriate. ET_{away} should equal the remainder of the 24-hour period spent away from the workplace. CA_{away} is generally assumed to equal zero, unless site-specific data suggest otherwise.

$$Industrial/Commercial\ EC_{24} = \frac{(CA_{work} \cdot ET_{work}) + (CA_{away} \cdot ET_{away})}{24\ hrs} \tag{4}$$

If multiple or variable microenvironments are present at a site, it is possible to use Equation 2 to generate a 24-hour TWA exposure concentration. However, consideration should be given to the use of portable sampling equipment to more accurately measure true exposure concentrations to the receptor(s) of concern over the entire exposure time, as opposed to stationary sampling equipment positioned in multiple areas where exposure occurs.

Non-cancer hazard quotients for heart defects can be derived using Equation 5, where HQ_{24} is the developmental hazard quotient; EC_{24} is the 24-hr time-weighted average exposure concentration calculated using Equations 2, 3, or 4; and the RfC is 2 $\mu g/m^3$. As shown in Equation 5, a hazard quotient is the ratio of the exposure to the non-cancer toxicity value. Thus, an HQ greater than 1 means that the exposure is greater than the RfC and exceeds a level of concern for that particular non-cancer health effect.

$$HQ_{24} = \frac{EC_{24}}{RfC} \tag{5}$$

Equation 5 can be combined with Equation 3 or 4 to calculate the developmental hazard quotients (HQ_{24}) for a residential or industrial/commercial receptor, as follows.

$$Residential\ HQ_{24} = \frac{CA_{res}}{2\frac{\mu g}{m^3}} \tag{6}$$

$$Industrial/Commercial\ HQ_{24} = \frac{(CA_{work} \cdot ET_{work}) + (CA_{away} \cdot ET_{away})}{24\ hrs \cdot 2\frac{\mu g}{m^3}} \tag{7}$$

Non-Cancer Hazard Quotients for Chronic Health Effects

Autoimmune disease, a co-critical endpoint upon which the TCE RfC is based, and kidney toxicity, the supporting endpoint, are both health effects associated with chronic or long-term exposures. Equation 8 is the standardized equation used to evaluate non-cancer hazard quotients for chronic health effects; the exposure parameters are defined in Table 1. If seasonal or temporal fluctuations in TCE concentrations potentially exist, consideration should be given as to whether sufficient data are available to generate an average concentration for use as the CA term. If the dataset is limited, it may be more health-protective to use the highest concentration detected.

$$HQ_{chronic} = \frac{CA\left(\frac{\mu g}{m^3}\right) \cdot ET\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF\left(\frac{days}{year}\right) \cdot ED(years)}{AT_{nc,chronic}(days) \cdot RfC\left(\frac{\mu g}{m^3}\right)} \tag{8}$$

The above equation can be presented in terms of residential or industrial/commercial exposure scenarios, as shown below. Note that it is only appropriate to calculate non-cancer hazard quotients for chronic health effects for those receptors with long-term exposures.

$$Residential\ HQ_{chronic} = \frac{CA_{res}\left(\frac{\mu g}{m^3}\right) \cdot ET_{res}\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF_{res}\left(\frac{days}{year}\right) \cdot ED_{child}(years)}{AT_{nc,chronic,child}(days) \cdot RfC\left(\frac{\mu g}{m^3}\right)} \tag{9}$$

$$Industrial/Commercial\ HQ_{chronic} = \frac{CA_{work}\left(\frac{\mu g}{m^3}\right) \cdot ET_{work}\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF_{work}\left(\frac{days}{year}\right) \cdot ED_{work}(years)}{AT_{nc,chronic,work}(days) \cdot RfC\left(\frac{\mu g}{m^3}\right)} \tag{10}$$

Cancer Risks

TCE is classified "carcinogenic to humans," based on kidney cancer, non-Hodgkin's lymphoma, and liver and biliary tract cancer. Equation 11 is the standardized equation used to evaluate excess individual lifetime cancer risks; the exposure parameters are defined in Table 1. If temporal fluctuations in TCE concentrations potentially exist, consideration should be given as to whether sufficient data are available to generate an average concentration for use as the CA term. If the dataset is limited, it may be more health-protective to use the highest concentration detected.

$$CR = \frac{CA\left(\frac{\mu g}{m^3}\right)\cdot ET\left(\frac{hrs}{day}\right)\cdot\left(\frac{1\,day}{24\,hrs}\right)\cdot EF\left(\frac{days}{year}\right)\cdot ED(years)\cdot IUR\left(\frac{\mu g}{m^3}\right)^{-1}}{AT_{cancer}(days)} \tag{11}$$

The above equation can be presented in terms of residential or industrial/commercial exposure scenarios, as shown below. Because a mutagenic mode of action has been established for kidney tumors associated with TCE, it is necessary to apply age-dependent adjustment factors when deriving risks for this cancer type in children (EPA, 2005b). ADAFs are not applied when deriving risks for non-Hodgkin's lymphoma or liver and biliary tract cancers associated with TCE exposures because they have not been determined to operate via a mutagenic mode of action. Because only adults are evaluated in an industrial/commercial exposure scenario and no adjustments for mutagenicity are made for adults (i.e., $ADAF_{adult} = 1$), ADAFs are not included in Equation 13.

$$Residential\ CR = \left(\frac{CA_{res}\left(\frac{\mu g}{m^3}\right)\cdot ET_{res}\left(\frac{hrs}{day}\right)\cdot\left(\frac{1\,day}{24\,hrs}\right)\cdot EF_{res}\left(\frac{days}{year}\right)}{AT_{cancer}(days)}\right)\cdot\Big[\left(ED_{0-2}(years)\cdot IUR_{kid}\left(\frac{\mu g}{m^3}\right)^{-1}\cdot ADAF_{0-2}\right)+\left(ED_{2-16}(years)\cdot IUR_{kid}\left(\frac{\mu g}{m^3}\right)^{-1}\cdot ADAF_{2-16}\right)+\left(ED_{16-26}(years)\cdot IUR_{kid}\left(\frac{\mu g}{m^3}\right)^{-1}\cdot ADAF_{adult}\right)+\left(ED_{res}(years)\cdot IUR_{N\&L}\left(\frac{\mu g}{m^3}\right)^{-1}\right)\Big] \tag{12}$$

$$Industrial/Commercial\ CR = \frac{CA_{work}\left(\frac{\mu g}{m^3}\right)\cdot ET_{work}\left(\frac{hrs}{day}\right)\cdot\left(\frac{1\,day}{24\,hrs}\right)\cdot EF_{work}\left(\frac{days}{year}\right)\cdot ED_{work}(years)\cdot IUR\left(\frac{\mu g}{m^3}\right)^{-1}}{AT_{cancer}(days)} \tag{13}$$

The definitions, values, and references for the exposure parameters and toxicity values used in this document are provided in Table 1. For the chronic scenarios, the EPA's standard default exposure parameters (EPA, 2014b) are used to best represent reasonable maximum exposure scenarios, which are the highest exposures reasonably expected to occur at a site (EPA, 1989). These values are based on the 2011 Exposure Factors Handbook (EPA, 2011c). Although the default exposure time for an indoor worker is 8 hours/day, it is preferable to identify a site-specific worker exposure time.

Table 1. Exposure Parameters and Toxicity Values.

Parameter	Definition	Units	Value	Reference
$ADAF_{0-2}$	Age-dependent adjustment factor – ages 0 to 2 years	-	10	EPA, 2005b
$ADAF_{2-16}$	Age-dependent adjustment factor – ages 2 to 16 years	-	3	EPA, 2005b
$ADAF_{adult}$	Age-dependent adjustment factor – ages 16 years and older	-	1	EPA, 2005b
AT_{24}	Averaging time – developmental effects	hours	24	-
AT_{cancer}	Averaging time – cancer	days	25,550	EPA, 2014b

Table 1. Exposure Parameters and Toxicity Values.				
Parameter	**Definition**	**Units**	**Value**	**Reference**
$AT_{nc,chronic,child}$	Averaging time – chronic non-cancer health effects, resident child	days	2,190	EPA, 2014b
$AT_{nc,chronic,work}$	Averaging time – chronic non-cancer health effects, indoor worker	days	9,125	EPA, 2014b
CA	Concentration of TCE in air	$\mu g/m^3$	Measured	-
CA_{res}	Concentration of TCE in air of the residence	$\mu g/m^3$	Measured	-
CA_{work}	Concentration of TCE in air of the workplace	$\mu g/m^3$	Measured	-
$ED_{0\text{-}2}$	Exposure duration – ages 0 to 2 years	years	2	EPA, 2005b
$ED_{2\text{-}16}$	Exposure duration – ages 2 to 16 years	years	14	EPA, 2005b
$ED_{16\text{-}26}$	Exposure duration – ages 16 to 26 years	years	10	EPA, 2005b
ED_{child}	Exposure duration – resident (child, ages 0 to 6 years)	years	6	EPA, 2014b
ED_{res}	Exposure duration – resident (child + adult, ages 0 to 26 years)	years	26	EPA, 2014b
ED_{work}	Exposure duration – indoor worker	years	25	EPA, 2014b
EF_{res}	Exposure frequency – resident	days/yr	350	EPA, 2014b
EF_{work}	Exposure frequency – indoor worker	days/yr	250	EPA, 2014b
ET_{away}	Exposure time – time spent away from work by an indoor worker (24 hrs/day minus ET_{work})	hrs/day	16 or site-specific	-
ET_{res}	Exposure time – time spent at home by a resident	hrs/day	24	EPA, 2014b
ET_{work}	Exposure time – time spent at work by an indoor worker	hrs/day	8 or site-specific	EPA, 2014b or site-specific
IUR	TCE inhalation unit risk - total	$(\mu g/m^3)^{-1}$	4.1E-06	EPA, 2011a
IUR_{kid}	TCE inhalation unit risk – kidney cancer	$(\mu g/m^3)^{-1}$	1.0E-06	EPA, 2011a
$IUR_{N\&L}$	TCE inhalation unit risk – non-Hodgkin's lymphoma and liver and biliary tract cancers	$(\mu g/m^3)^{-1}$	3.1E-06	EPA, 2011a
RfC	TCE reference concentration	$\mu g/m^3$	2	EPA, 2011a
THQ	Target hazard quotient	-	1	-
TR	Target cancer risk	-	1E-04	Upper-end of Target Cancer Risk Range

Action Levels

Level of Concern for Developmental Effects

Equations 2 and 5 can be manipulated to solve for the level of concern for developmental health effects, using a target non-cancer hazard quotient of 1, as follows. Note that the only exposure parameter that can vary in this calculation is the exposure time. The TCE levels of concern for developmental effects based on standard exposure times are provided in Table 2. For a 24-hour residential scenario, the developmental LOC equals 2 $\mu g/m^3$. For a typical 8-hour industrial/commercial scenario, the developmental LOC equals 6 $\mu g/m^3$. Site-specific developmental LOCs may be derived using alternate exposure times; for example, a 10-hour exposure time results in a developmental LOC of 4.8 $\mu g/m^3$.

$$TCE\ LOC_{developmental}\left(\frac{\mu g}{m^3}\right) = \frac{THQ \cdot AT_{24}(hrs) \cdot RfC\left(\frac{\mu g}{m^3}\right)}{ET\left(\frac{hrs}{day}\right)} \tag{14}$$

$$TCE\ Residential\ LOC_{developmental}\left(\frac{\mu g}{m^3}\right) = \frac{1 \cdot 24\ hrs \cdot 2\ \frac{\mu g}{m^3}}{24\ hrs} \tag{15}$$

$$TCE\ Industrial/Commercial\ LOC_{developmental}\left(\frac{\mu g}{m^3}\right) = \frac{1 \cdot 24\ hrs \cdot 2\ \frac{\mu g}{m^3}}{ET_{work}\left(\frac{hrs}{day}\right)} \tag{16}$$

Level of Concern for Chronic Non-Cancer Health Effects

Equation 8 can be manipulated to solve for the level of concern for chronic, non-cancer health effects, using a target non-cancer hazard quotient of 1 and the exposure parameters presented in Table 1, as follows. For a residential scenario, this LOC equals 2.1 μg/m³, which is the value listed as the non-cancer residential air Regional Screening Level for TCE, based on an HQ of 1 (EPA, 2016b). For an industrial/commercial scenario, the chronic LOC equals 8.8 μg/m³, which is the value listed as the non-cancer worker air RSL for TCE, based on an HQ of 1. Site-specific chronic LOCs may be derived using alternate exposure times or other parameters.

$$TCE\ LOC_{chronic}\left(\frac{\mu g}{m^3}\right) = \frac{THQ \cdot AT_{nc,chronic}(days) \cdot RfC\left(\frac{\mu g}{m^3}\right)}{ET\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF\left(\frac{days}{year}\right) \cdot ED(years)} \tag{17}$$

$$TCE\ Residential\ LOC_{chronic}\left(\frac{\mu g}{m^3}\right) = \frac{THQ \cdot AT_{nc,chronic,child}(days) \cdot RfC\left(\frac{\mu g}{m^3}\right)}{ET_{res}\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF_{res}\left(\frac{days}{year}\right) \cdot ED_{child}(years)} \tag{18}$$

$$TCE\ Industrial/Commercial\ LOC_{chronic}\left(\frac{\mu g}{m^3}\right) = \frac{THQ \cdot AT_{nc,chronic,work}(days) \cdot RfC\left(\frac{\mu g}{m^3}\right)}{ET_{work}\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF_{work}\left(\frac{days}{year}\right) \cdot ED_{work}(years)} \tag{19}$$

Level of Concern for Cancer Risks

Equations 11, 12, and 13 can be manipulated to solve for the level of concern for cancer risks, using a target excess cancer risk (TR) of 1E-04, which is the upper bound of the EPA's target cancer risk range, and the exposure parameters presented in Table 1, as follows. For a residential scenario, this LOC equals 48 μg/m³, and for an industrial/commercial scenario, the cancer LOC equals 300 μg/m³.

$$TCE\ LOC_{cancer}\left(\frac{\mu g}{m^3}\right) = \frac{TR \cdot AT_{cancer}(days)}{ET\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF\left(\frac{days}{year}\right) \cdot ED(years) \cdot IUR\left(\frac{\mu g}{m^3}\right)^{-1}} \tag{20}$$

$$TCE\ Residential\ LOC_{cancer}\left(\frac{\mu g}{m^3}\right) =$$

$$\frac{TR \cdot AT_{cancer}(days)}{ET\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF\left(\frac{days}{year}\right) \cdot \left[\begin{array}{c}\left(ED_{0-2}(years) \cdot IUR_{kid}\left(\frac{\mu g}{m^3}\right)^{-1} \cdot ADAF_{0-2}\right) + \left(ED_{2-16}(years) \cdot IUR_{kid}\left(\frac{\mu g}{m^3}\right)^{-1} \cdot ADAF_{2-16}\right) + \\ \left(ED_{16-26}(years) \cdot IUR_{kid}\left(\frac{\mu g}{m^3}\right)^{-1} \cdot ADAF_{16-26}\right) + \left(ED_{res}(years) \cdot IUR_{N\&L}\left(\frac{\mu g}{m^3}\right)^{-1}\right)\end{array}\right]} \tag{21}$$

$$TCE\ Industrial/Comm.\ LOC_{cancer}\left(\frac{\mu g}{m^3}\right) = \frac{TR \cdot AT_{cancer}(days)}{ET_{work}\left(\frac{hrs}{day}\right) \cdot \left(\frac{1\ day}{24\ hrs}\right) \cdot EF_{work}\left(\frac{days}{year}\right) \cdot ED_{work}(years) \cdot IUR\left(\frac{\mu g}{m^3}\right)^{-1}} \tag{22}$$

As shown below in Table 2, the levels of concern for developmental health effects are lower than the LOCs for chronic health effects and cancer, for both residential and occupational scenarios, when based on target hazard quotients of 1 or target cancer risks of 1E-04. These are the levels of risk that, when exceeded, warrant action under the National Contingency Plan. Basing the Region 7 TCE action levels on the developmental LOCs is protective for all potential forms of adverse health effects associated with TCE. Thus, the action level for a residential scenario is 2 μg/m^3, and the action level for a typical industrial/commercial scenario with an 8-hr workday is 6 μg/m^3. As previously mentioned, the developmental LOC, and thus the action level, is highly dependent on the exposure time. Therefore, for non-residential exposure scenarios, careful consideration should be given to the value selected as the exposure time.

Table 2. Levels of Health Concern for Trichloroethylene (μg/m^3), THQ = 1 and TR = 1E-04.	
Residents (24-hr Exposure Scenario)	
Developmental Non-Cancer LOC:	*2*
Chronic Non-Cancer LOC:	*2.1*
Cancer LOC:	*48*
Region 7 Residential TCE Action Level:	**2**
Industrial/Commercial Workers (8-hr Exposure Scenario)	
Developmental Non-Cancer LOC:	*6*
Chronic Non-Cancer LOC:	*8.8*
Cancer LOC:	*300*
Region 7 Industrial/Commercial TCE Action Level:	**6**

Risk Management Considerations

If the TCE action level is exceeded, this indicates a potential imminent threat to human health, and early or interim action(s) should be taken to eliminate, reduce, and/or control the hazards posed by the site (EPA, 2014d). At Superfund sites, coordination between the remedial and removal programs should immediately commence as early as the receipt of preliminary sampling results indicative of a potential human health concern (EPA, 2016c). Potential receptors should be informed of the results and potential risks to human health. Standard Region 7 practice is to communicate this information via data transmittal letters submitted to property owners and employers, but when TCE action levels are exceeded, tenants, residents, employees and others who may be exposed should also be informed. Although the action levels derived in this document are applicable to women in the first trimester of pregnancy, note that the levels protective of autoimmune disease and kidney toxicity in all individuals are not significantly different, at 2.1 and 8.8 μg/m^3, for residents and workers, respectively. Depending on the concentrations detected, immediate site actions could include relocation, restricting the time residents or workers remain in areas exceeding action levels, opening basement or lower level windows for ventilation (using a fan), sealing cracks in the slab, sealing sump pits, sealing cinder block or stone walls, and/or using air filtration systems. Vapor mitigation systems or adjustments to HVAC systems may be used to minimize exposures on a more long-term basis. Post-remedy testing and continued operation and maintenance is necessary to ensure protection of human health until the source of TCE in soil and/or groundwater is ultimately addressed.

Other EPA Regions and states have derived tiered action levels prescribing the types and urgency of various responses, as described below.

- Although Region 7 consistently uses a THQ of 1 as the basis for both removal and remedial Superfund actions, other Regions have used a THQ of 3 as a science policy approach to prioritize actions that may warrant the use of removal authority, with ultimate cleanup goals based on a THQ of 1. Since non-cancer toxicity values have historically been based on effects resulting from chronic exposure, this practice assumes that the most highly contaminated sites will be remediated first, but all sites will be remediated before exposures have occurred for a sufficiently long duration (e.g., 25 years as a worker or 26 years as a resident) to pose significant health risks.

This assumption is not protective of the short-term health effects associated with TCE, in which the critical window of susceptibility is an approximate three week period and a single exposure during this critical time may result in cardiac malformations.

- Tiered action levels could also be derived by reducing the uncertainty factor applied to the RfC from 10 to 1. The existing UF of 10 is applied for uncertainty regarding differences in pharmacodynamics between animals and humans and between the general population and sensitive subpopulation. Other than the toxicokinetic variability characterized by the physiologically-based pharmacokinetic model, EPA (2011a) indicates that there are inadequate chemical-specific data to quantify the degree of differential susceptibility due to factors such as genetic polymorphisms, race/ethnicity, preexisting health status, lifestyle factors, and nutritional status. The UF of 10 was included in the extensive peer-review process described in this document, and Region 7 does not have justification to alter this value.
- Similarly, the selection of a 1% excess risk as the benchmark response and a human equivalent concentration for a toxicokinetically sensitive individual at the 99th percentile were both extensively reviewed, and Region 7 does not have justification to alter these criteria.

Although Region 7 has not developed tiered levels because this approach may not be protective of human health, higher concentrations of TCE are associated with greater health risks. Actions should be implemented as quickly as is practicable to minimize risks of developmental toxicity. This document reinforces that Region 7 should expedite actions to protect human health whenever the TCE air concentration exceeds 2 $\mu g/m^3$ in a residential scenario or 6 $\mu g/m^3$ for an 8-hour worker scenario.

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